AMENDMENTS

Listing of Claims:

The following listing of claims replaces all previous listings or versions thereof:

1. (Original) A method for evaluating the potential efficacy of an EGFR-targeting therapeutic agent for the treatment of cancer in a patient comprising determining the sequence of a polymorphism in one or both EGFR genes in the patient.

2. (Original) The method of claim 1, wherein the polymorphism is at, or in linkage disequilibrium with, a nucleotide position selected from the group consisting of nucleotide positions -1435, -1300, -1249, -1227, -761, -650, -544, -486, -216, -191, 169, and 2034.

3. (Original) The method of claim 2, wherein the polymorphism is, or is in linkage disequilibrium with, a polymorphism selected from the consisting of –1435 C>T, -1300 G>A, -1249 G>A, -1227 G>A, -761 C>A, -650 G>A, -544 G>A, -486 C>A, -216 G>T, -191 C>A, 169 G>T, and 2034 G>A.

4. (Original) The method of claim 1, further comprising determining the sequence of at least two polymorphisms in one or both EGFR genes in the patient.

5. (Original) The method of claim 1, wherein the EGFR-targeting therapeutic agent is an EGFR-tyrosine kinase inhibitor.

6. (Original) The method of claim 5, wherein the EGFR-tyrosine kinase inhibitor is gefitinib or erlotinib.

7. (Original) The method of claim 1, wherein the EGFR-targeting therapeutic agent is a monoclonal antibody.

8. (Original) The method of claim 7, wherein the monoclonal antibody is cetuximab.

9. (Cancelled)

10. (Currently Amended) The method of claim [[9]] 8, wherein a T at position –216 on an allele is an indicator of higher expression of EGFR protein, and further wherein the higher expression of EGFR protein is an indicator of decreased efficacy of the EGFR-targeting therapeutic agent.

11. (Original) The method of claim 1, further comprising determining the sequence of a polymorphism in both EGFR genes in the patient.

12. (Previously presented) The method of claim 1, wherein determining the sequence of a polymorphism is performed by a hybridization assay , by an allele specific amplification assay, by a sequencing or a microsequencing assay, or by digestion with a restriction enzyme.

13-15. (Cancelled)

16. (Original) The method of claim 1, further comprising obtaining a sample.

17. (Original) The method of claim 16, wherein the sample comprises buccal cells, mononuclear cells, or cancer cells.

18. (Original) The method of claim 1, further comprising administering the EGFR-targeting therapeutic agent to the patient.

19. (Original) A method for predicting the clinical prognosis for a cancer patient comprising determining the sequence of a polymorphism in one or both EGFR genes in the patient.

20. (Original) The method of claim 19, further comprising determining the sequence of a polymorphism in both EGFR genes in the patient.

21. (Original) The method of claim 19, wherein the polymorphism is at, or in linkage disequilibrium with, a nucleotide position selected from the group consisting of nucleotide positions -1435, -1300, -1249, -1227, -761, -650, -544, -486, -216, -191, 169, and 2034.

22. (Original) The method of claim 21, wherein the polymorphism is, or is in linkage disequilibrium with, a polymorphism selected from the consisting of –1435 C>T, -1300 G>A, -1249 G>A, -1227 G>A, -761 C>A, -650 G>A, -544 G>A, -486 C>A, -216 G>T, -191 C>A, 169 G>T, and 2034 G>A.

23. (Cancelled)

24. (Previously presented) The method of claim 19, wherein a T at position –216 on an allele is an indicator of an increased expression of EGFR protein.

25. (Original) The method of claim 24, wherein the increased expression of EGFR protein is predictive of poor prognosis.

26. (Original) The method of claim 25, wherein the poor prognosis indicates increased resistance to chemotherapy, hormonal therapy, or radiotherapy.

27. (Original) The method of claim 25, wherein the poor prognosis indicates increased risk of metastasis.

28. (Original) A method for evaluating a patient's risk of toxicity to an EGFR-targeting therapeutic agent comprising determining the sequence of a polymorphism in one or both EGFR genes in the patient.

29. (Original) The method of claim 28, wherein the polymorphism is at, or in linkage disequilibrium with, a nucleotide position selected from the group consisting of nucleotide positions -1435, -1300, -1249, -1227, -761, -650, -544, -486, -216, -191, 169, and 2034.

30. (Original) The method of claim 29, wherein the polymorphism is, or is in linkage disequilibrium with, a polymorphism selected from the consisting of –1435 C>T, -1300 G>A, -1249 G>A, -1227 G>A, -761 C>A, -650 G>A, -544 G>A, -486 C>A, -216 G>T, -191 C>A, 169 G>T, and 2034 G>A.

31. (Cancelled)

32. (Previously presented) The method of claim 30, wherein a T at position –216 on one or both alleles is an indicator of decreased toxicity of the EGFR-targeting therapeutic agent.

33. (Original) The method of claim 28, further comprising determining the sequence of a polymorphism in both EGFR genes in the patient.

34-35. (Cancelled)

36. (Withdrawn) A kit for evaluating the potential efficacy of an EGFR-targeting therapeutic agent in a patient comprising a nucleic acid for determining the sequence of a polymorphism in an EGFR gene locus or a restriction enzyme for determining the sequence of a polymorphism in an EGFR gene locus.

37. (Withdrawn) The kit of claim 36, wherein the nucleic acid is a primer for amplifying a polymorphism at a nucleotide position selected from the group consisting of -1435, -1300, -1249, -1227, -761, -650, -544, -486, -216, -191, 169, and 2034.

38. (Withdrawn) The kit of claim 36, wherein the nucleic acid is a specific hybridization probe designed to detect a polymorphism at a nucleotide position selected from the group consisting of -1435, -1300, -1249, -1227, -761, -650, -544, -486, -216, -191, 169, and 2034.

39. (Withdrawn) The kit of claim 38, wherein the specific hybridization probe is comprised in an oligonucleotide array or microarray.

40. (Cancelled)